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## Bacterial left atrial mural thrombosis associated with uraemic nephritis in a dog - a case report

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### ABSTRACT

Necropsy examination of a five-year-old female German shepherd dog revealed a pale greyish-white thrombotic mass adherent to the lateral wall of left atrium of heart. Liver was enlarged, mottled and showed multifocal pale foci. Both kidneys were small, pale with granular cortical surface. Cultural examination of heart blood swab revealed the presence of *Staphylococcus aureus*. Clinical laboratory examination revealed elevation of blood urea nitrogen (312 mg/dL) and creatinine (19.7 mg/dL). Microscopical examination showed thrombotic mass adhering to the endocardial surface, which contained blood cell debris and numerous coccoid bacterial clumps. Blood vessels adjacent to the thrombus in the myocardium revealed mineralisation of the tunica intima with perivascular lympho-plasmacytic infiltration. Thus, a case of left atrial vegetative endocarditis developed from *S. aureus* infection due to uraemic endocardial injury is reported.

**Key words:** left atrial mural thrombosis, uraemia, *Staphylococcus aureus*, dog

### Introduction

Endocarditis is mostly seen in valves in dogs (JONES and HUNT, 1983; SISSON and THOMAS, 1984; COOK et al., 2005). Such lesions are not frequently seen in dogs in India. A very rare case of left atrial mural thrombosis encountered in a dog is reported.

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## Materials and methods

A carcass of five-year-old German shepherd dog was presented to the Department of Veterinary Pathology, Madras Veterinary College, Chennai-600 007 India, for routine post-mortem examination. Heart blood swab collected was subjected to bacteriological examination (HOLT et al., 1994). Serum was collected for biochemical analysis of blood urea nitrogen (BUN), creatinine (BALISTREI and ROBERTREJ, 1996) and alanine aminotransferase-ALAT (WHELTON et al., 1996). Tissue pieces collected from heart, liver, lung, kidneys and spleen were immediately fixed in 10 per cent formal saline and processed by routine procedures. Thin sections of about 5  $\mu$ m thickness cut were stained with haematoxylin and eosin.

## Results

External examination of carcass revealed a circular wound of about 0.5 cm in diameter on accessory digit, above the second digit of left hind limb. Oral examination revealed two irregular ulcers of about 0.5 cm in diameter on the mucosa at the angle of mouth. Necropsy revealed the presence of a pale greyish-white thrombus mass of about 0.5 cm in diameter adhering to the lateral wall of left atrium of the heart (Fig. 1). Both lungs were oedematous and congested. The visceral surface of ventral end of spleen showed patchy areas of subcapsular haemorrhages. Liver was enlarged and mottled and showed multifocal pale foci of 2 to 5 mm in diameter on the parietal surface, which on incision were found to be extended into the parenchyma. Both kidneys were small and shrunken, with capsules adherent to cortical surfaces. Cortical surfaces were pale and granular in appearance.



Fig. 1. Heart-Pale greyish-white thrombus mass adhering to the wall of left atrium

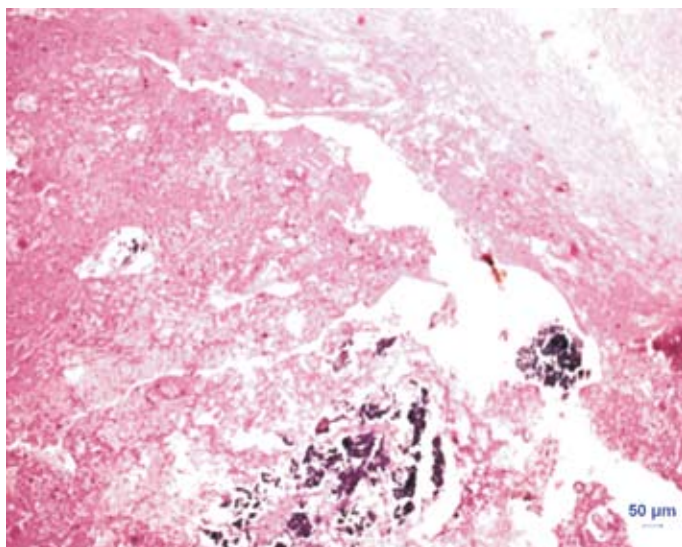


Fig. 2. Heart showing thrombus mass adhering to the endocardial surface. H&E; scale bar = 50 µm.

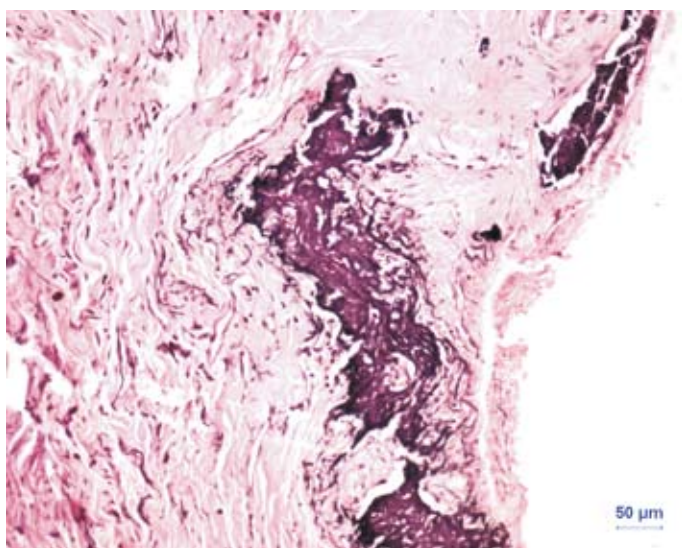


Fig. 3. Heart. Myocardial blood vessels showing mineralization. H&E stain; scale bar = 50 µm.

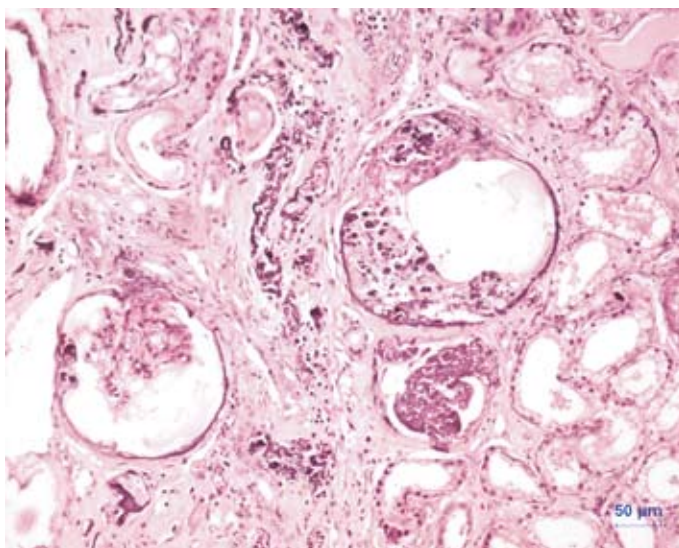


Fig. 4. Kidney. Glomeruli showing atrophy, calcification of Bowman's membrane and capillary tufts and faintly eosinophilic fluid and tubular calcification. H&E; scale bar = 50 µm.

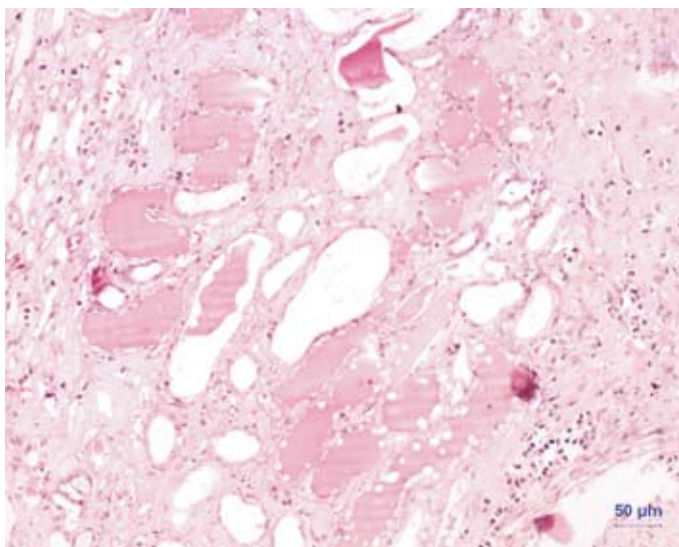


Fig. 5. Kidney. Tubules showing degeneration and necrosis of epithelium with calcification of basement membrane. H&E; scale bar = 50 µm.

A creamy-white content mixed with thick sparse mucus was seen in the stomach. Intestinal contents were viscous, light yellow and mixed with mucus. Jejunal mucosa showed two patchy areas of haemorrhages. Urinary bladder was thickened, with diffuse areas of haemorrhages on the mucosa.

Microscopical examination of heart revealed thrombotic mass adhering to the endocardial surfaces (Fig. 2). The thrombotic mass contained blood cells, debris and numerous coccoid bacterial clumps. Areas of calcification were also evident in the thrombus mass. Fibrovascular tissue with infiltrating mononuclear cells beneath the thrombus was seen extending into the myocardium. Blood vessels adjacent to the thrombus in the myocardium revealed mineralisation of the tunica intima, with perivascular lymphoplasmocytic infiltration (Fig. 3).

Kidneys revealed multifocal areas of inflammatory infiltrates consisting of predominantly mononuclear cells and fibroplasia, with a moderate amount of collagen in the cortex. Many of the glomeruli showed atrophy, calcification of Bowman's membrane and capillary tufts, and accumulation of faintly eosinophilic exudate. Interstitium was very prominent, with scattered infiltration of mononuclear cells consisting of predominantly plasma cells. Fibroplastic proliferation and collagen deposits were seen in the interstitium. Many of the tubules showed degenerative and necrotic changes in the lining epithelium. Tubules also showed calcification of basement membrane, and a few tubules contained calcified mass completely obstructing the lumen (Fig. 4 and 5). A few tubules showed marked distension containing faintly eosinophilic exudates with cell debris. Interstitium in the medulla also showed collagen deposition, scattered infiltration of mononuclear cells and multifocal areas of prominent distension of tubules containing eosinophilic fluid.

Liver showed chronic venous congestion with widening of sinusoids. Peripheral hepatocytes showed vacuolar degenerative changes and focal areas of degenerative changes around the central vein. Lungs showed oedema and emphysema. Calcification of intima was observed in splenic arteries.

Bacteriological examination of heart blood swab revealed the presence of *Staphylococcus aureus*, which was confirmed by biochemical characters (HOLT et al., 1994). Clinical laboratory findings showed elevation of blood urea nitrogen (312 mg/dL), creatinine (19.7 mg/dL) and ALAT (101.3 IU).

## Discussion

Left atrial mural thrombosis was confirmed by gross and histopathological examination. Retrospective study showed that only two cases out of 1436 necropsies conducted on canines during the last decade in our department had vegetative endocarditis, of which one involved atrioventricular valve and another, semilunar valves. This indicated

a rare occurrence of vegetative endocarditis in dogs. Valvular endocarditis is common in dogs (JONES and HUNT, 1983; Sisson and THOMAS, 1984; COOK et al., 2005) while mural endocarditis was observed in the present case. This constitutes another rarity.

The *S. aureus*, incriminated as a cause of vegetative endocarditis (CALVERT, 1982), could have entered into body through the wound on the accessory digit and above the second digit of left hind limb, which was in accordance with a previous report (JONES and HUNT, 1983). History revealed that it was resistant to several antibiotic treatments given over a period of one-and-a-half months. The *S. aureus* organism was reported to be resistant to varieties of antibiotics by earlier workers (JONES and HUNT 1983). In spite of prolonged treatment with antibiotics, the *S. aureus* could be lodged in the wall of endocardium, producing thrombus in the lateral wall of lateral atrium. *S. aureus* has been known to produce endocarditis in cattle (JONES and HUNT, 1983).

Elevation of BUN indicated uraemia (COLES, 1986). Uraemia is the most common cause of endocarditis in dogs, followed by endocardial necrosis and thrombosis (JONES and HUNT, 1983). The most common form of mural endocarditis is that occurring in renal failure in dogs and lesions confined to the left atrium (ROBINSON and MAXIE, 1993). In the present study, lesions were seen in the left atrium in association with uraemia. Endocardial and major arterial lesions are more common in acute than in chronic renal failure (ROBINSON and MAXIE, 1993), while we have recorded lesions in chronic nephritis with uraemia. In uraemia, mineralization was reported in glomeruli and small blood vessels (MAXIE and PRESCOTT, 1993) as we have recorded in the Bowman's membrane and capillary tufts of glomeruli and splenic arteries.

The left atrial mural thrombosis might have occurred due to uraemic endocarditis followed by septicaemia involving *S. aureus*.

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**PAZHANIVEL, N., G. V. S. RAO, T. G. PRABAKAR, V. T. GEORGE, C. BALACHANDRAN, B. M. MANOHAR: Povezanost bakterijske tromboze stijenke lijeve srčane pretkljetke i uremičnog nefritisa u psa - prikaz slučaja. Vet. arhiv 77, 87-93, 2007.**

**SAŽETAK**

Razudbom petogodišnje ženke, pasmine njemački ovčar, ustanovljena je blijeda sivkasto bijela nakupina vezana uz lateralnu stijenku lijeve srčane pretkljetke. Jetra su bila povećana, prošarana mnogobrojnim blijedim žarištima. Oba bubrega bila su mala, blijeda s granularnom površinom kore. Pretragom srčane krvi dokazana je prisutnost bakterije *Staphylococcus aureus*. Kliničkom laboratorijskom pretragom ustanovljena je povećana koncentracija dušika u krvi (312 mg/dL) i kreatinina (19,7 mg/dL). Mikroskopska pretraga pokazala je nakupinu tromba prilijepljenu na površinu endokarda, koja je sadržavala ostatke krvnih stanica i mnoge nakupine kokoidnih bakterija. Krvne žile miokarda koje prileže uz tromb pokazale su mineralizaciju unutarnje ovojnice s perivaskularnom limfoplazmatičnom infiltracijom. Na temelju navedenoga zaključuje se da je riječ o vegetativnom endokarditisu lijeve srčane pretkljetke, razvijenom nakon zaraze bakterijom *S. aureus* povezane s uremičnom endokardijalnom ozljedom.

**Ključne riječi:** tromboza, lijeva pretkljetka, uremija, *Staphylococcus aureus*, pas

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